Division of Lung Diseases and
Office of Prevention, Education, and Control

BRONCHOPULMONARY DYSPLASIA

NATIONAL INSTITUTES OF HEALTH
NATIONAL HEART, LUNG, AND BLOOD INSTITUTE

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Coordinated by: Office of Prevention, Education, and Control

The NHLBI anniversary logo represents 50 years of success; people doing science to improve the health of people.

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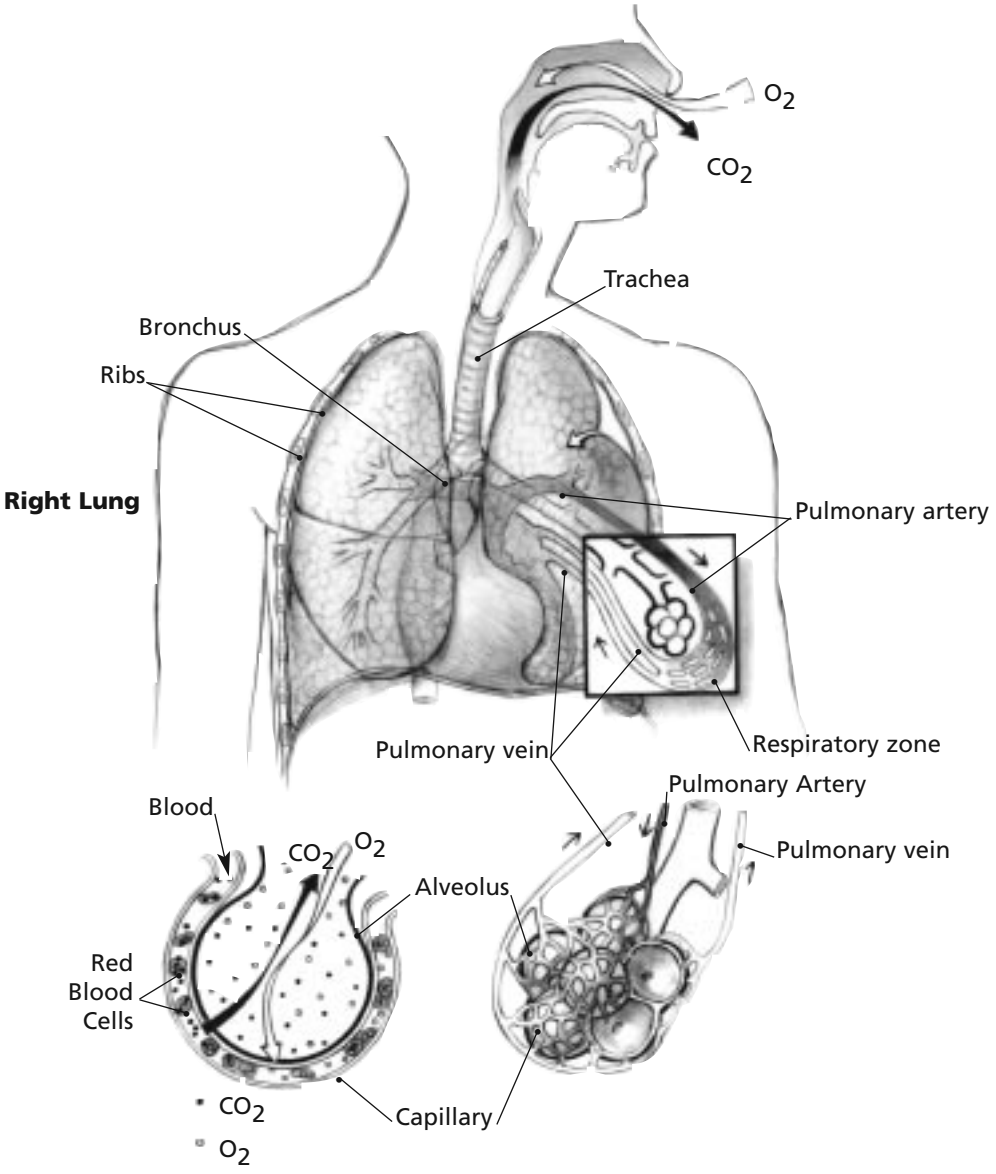


Lung Structure and Gas Exchange

Inhaled air travels through the airways to the alveoli. Blood is pumped out of the heart through the pulmonary arteries to a network of capillaries that surround the alveoli. The oxygen of the inhaled air diffuses out of the alveoli into the blood while carbon dioxide in the blood moves into the alveoli to be exhaled. The oxygen-rich blood is returned to the heart through the pulmonary veins.

Introduction

Your baby has been diagnosed with *bronchopulmonary dysplasia* (BPD). BPD is a *chronic* lung disease of newborn infants marked by *inflammation* of the airways. The lungs of babies with BPD are immature or have not developed normally. Their lungs are therefore unable to perform *gas exchange*. Gas exchange is the primary function of the lungs. This term is used for the process by which lungs transfer *oxygen* from inhaled air into the bloodstream in exchange for carbon dioxide from the blood to be exhaled. (see illustration on page i) Inhaled air travels to the *alveoli* (tiny air sacs located at the end of the conducting airways of the lungs) where it comes in contact with blood containing carbon dioxide. The blood travels through a fine network of *pulmonary* vessels called *capillaries*. Gas exchange occurs instantaneously in normal infants as the red blood cells pass through the capillaries. It does not occur adequately in BPD infants because their alveoli and capillaries are not fully developed.

This booklet explains what BPD is, its signs and symptoms, how it is treated, and what parents might expect as an infant with a history of BPD grows. Words that may not be familiar are in italic and are explained when they first appear; they also are defined in the glossary at the end of the booklet. The centerfold presents a more technical discussion of BPD. Also in the centerfold is a description of highlights of the National Heart, Lung, and Blood Institute's research programs in BPD.

What Is BPD?

BPD is a serious, chronic lung disease of infants. The dictionary defines BPD as abnormal development or growth (dysplasia) of the lungs and air passages.

BPD develops most commonly during the first 4 weeks after birth.

BPD was first described in 1967 by William Northway, a radiologist at Stanford University, as a chronic lung disease that occurred in premature babies who needed intensive oxygen therapy to survive *respiratory distress syndrome* (RDS) (see sidebar on page 3). Northway noted that the *symptoms* and chest x-rays of these babies were different than those seen in newborns with other lung diseases.

BPD develops most commonly during the first 4 weeks after birth. Although it is seen most often in premature babies, it can also occur in full-term babies who have respiratory problems during their first days of life. Babies who are still dependent on a respirator for oxygen at 28 days of age and whose chest x-rays are typical of BPD are considered to have the disorder.

BPD can occur when a baby's lungs which have not fully developed at birth have to begin breathing immediately and also adjust to adverse conditions outside the mother's womb. Among the adverse conditions which injure the lungs and cause BPD are oxygen under high pressure and infectious agents such as bacteria or viruses.

How Common Is BPD?

BPD is a worldwide problem. BPD and RDS together are probably responsible for most of the infant morbidity and mortality in developed countries. BPD ranks with *cystic fibrosis* and *asthma* among the most common chronic lung diseases in infants in the United States. Approximately 5,000 to 10,000 new cases of BPD (20 to 30 percent of infants surviving RDS) occur each year.

The Role of RDS in the Development of BPD

RDS is a lung disease that occurs in premature infants. It is also called *hyaline membrane disease* (HMD). RDS occurs because the lungs of premature babies produce too little *surfactant*, the critical substance that coats the inner surface of the lungs of full-term babies.

Within 3 to 4 hours after birth of these premature babies, proteins from the blood and from the *amniotic fluid* that remains in the newborn's lungs begin to leak into the gas exchange structures (terminal *bronchioles* and alveolar *ducts*) of the baby's lungs. These proteins and dead blood *cells* form a thick membranous layer called a hyaline *membrane*. The hyaline membrane becomes a physical barrier that prevents the entry of oxygen into the lungs. The formation of the hyaline membrane is complete within 12 to 24 hours.

The lungs of most full-term infants are soft and pliable. They stretch and contract with each breath. By contrast, the lung tissue of RDS babies is stiff and these newborns must work very hard to breathe. Symptoms of RDS include grunting noises with each breath, very rapid breathing (tachypnea), poor gas exchange, periods in which breathing stops for 20 seconds or longer (apnea), abnormal contraction of the muscles between the ribs and between the breastbone (the sternum) and the chest wall, and a blue tinge in the skin around the lips and in the nail beds (*cyanosis*) that indicates lack of oxygen. Most RDS babies develop *respiratory failure* and they need mechanical breathing assistance from a *ventilator* (respirator) to survive. Some of the babies who survive RDS go on to develop BPD.

In the early years after BPD was first reported, the treatment for RDS usually consisted of *positive pressure ventilation*, using a respirator to give high levels of oxygen under high pressure. Today surfactant replacement therapy is the standard treatment and it has excellent results.

Development of BPD is not limited to RDS survivors. BPD can also occur in full-term babies.

Development of BPD is not limited to RDS survivors. Any newborn infant who has serious respiratory problems in its first few days after birth is at risk of developing BPD. Although BPD is most common in premature babies, it can occur in full-term infants who need mechanical *ventilation* and oxygen under pressure for problems such as neonatal *pulmonary hypertension*.

Ninety percent of the infants who develop BPD are premature and weigh less than 1500 grams (3.5 pounds). In very premature infants (weighing 1 to 1.5 pounds or born after less than 22 weeks of gestation) BPD sometimes develops even in the absence of *acute* respiratory problems.

The risk of BPD increases with decreasing birth weight and *gestation period*. BPD occurs in 5 percent of infants whose birth weight is over 1,500 grams; the incidence rises to 85 percent in surviving newborns weighing between 500 and 700 grams (1 to 1.5 pounds). Male gender and non-African ethnicity seem to be additional *risk factors*. *Genetic* factors also may have a role.

Infant with BPD in an incubator

In the late 1960s, infants with BPD who survived past 4 weeks of age were an average of 6 weeks premature and their average weight was 2,234 grams (a little more than 4.5 pounds). Improved and more sophisticated neonatal critical care now makes it possible for the majority of infants weighing at least 500 grams to survive. This increased survival of very low birth weight infants is a major factor contributing to the growing incidence of BPD.

What Causes BPD?

BPD does not develop in all infants for the same reason. When it was first described, doctors thought that BPD was a result of lung injury from the *mechanical ventilation* and supplemental oxygen provided as therapy for RDS.

BPD does not develop in all infants for the same reason.

Today the specialists who treat BPD believe that, although RDS and premature birth play a role in the development of the disorder, these are not the only factors. Rather, BPD appears to reflect the limited ability of the baby's lungs during its first hours and days after birth to respond to adverse situations. These challenges may include oxygen toxicity, mechanical lung trauma, infections, or *pneumonia*. The state of immaturity of the lung at birth and the type of lung injury probably determine how the newborn's lungs respond and whether or not BPD actually develops.

What Are the Signs And Symptoms of BPD?

The signs and symptoms of BPD and how severe they are vary from infant to infant. They reflect differences in lung maturity and in the severity of disease. Respiratory signs include:

Signs and symptoms of BPD can vary from infant to infant.

- rapid shallow breathing (tachypnea), sucked-in ribs and chest (retraction), and cough;
- movement of the chest and abdomen in opposite directions with every breath (paradoxical or see-saw *respiration*); and
- *wheezing*.

The BPD infant's struggle to breathe is reflected in an abnormal posture of its neck, shoulders, and trunk. These babies also crane their necks as they use their neck muscles to try to get as much air as possible into their lungs.

Many of the symptoms of BPD are seen with other breathing problems, for example, severe asthma. If an infant shows any of these symptoms, the doctor will conduct tests to find the cause.

How is BPD Diagnosed?

Although BPD may begin as early as 1 week of age, it is difficult to diagnose until a baby is 14 to 30 days old. A diagnosis of BPD is based on:

Need for supplemental oxygen beyond 28 days and a typical x-ray confirm diagnosis of BPD.

- a history of lung injury in the first days after birth, (Pulmonary injury can result when a respirator must be used to provide oxygen under pressure for a minimum of 3 days during the first 2 weeks of life.)
- a continuing need for supplemental oxygen at age 28 days, and
- persistence of the clinical signs of respiratory difficulty beyond 28 days of age.

An x-ray of the infant's chest is also taken to help diagnose BPD. However, the most important functional criterion for the diagnosis of BPD is the need for supplemental oxygen beyond the 28th day of life.

The criteria used for a diagnosis of BPD vary among *neonatologists*. They include how long respiratory distress exists and how long the baby needs to be on a respirator. Many doctors make a diagnosis of BPD in the second or third week of life. However, some doctors defer a diagnosis until the baby is at least 28 days old.

What is the Outcome for Babies with BPD?

Most infants over 1,500 grams birth weight (3.5 pounds) who develop BPD have severe respiratory failure in the first week of life which may continue for several weeks. Extremely premature infants (those weighing less than 1,500 grams at birth, and especially those weighing less than 1,000 grams), seem to have minimal lung disease or acute lung disease that has apparently resolved, and then symptoms of BPD begin in the second week of life.

As BPD develops and progresses, the infants become increasingly dependent on oxygen and artificial ventilation. They typically display recurrent blueing or cyanotic episodes, and asthma-like symptoms. They may develop life-threatening *bronchiolitis* and other pulmonary complications. They may also develop serious medical complications of the heart, kidney, gastrointestinal tract, brain, or *retina*. In severe cases, the baby may die. Most of these deaths occur during the baby's first hospital stay. They are due to *progressive* respiratory failure, or its complications.

Most BPD infants will show continued slow improvement, but some may require extra weeks and months of care in the neonatal intensive care unit (NICU). It is estimated that infants with BPD require intensive in-hospital care for an average of 120 days.

At 36 weeks after conception (4 weeks before the baby's original due date), nearly a third of the infants with BPD no longer require supplemental oxygen therapy. Those who continue to require supplemental oxygen are usually otherwise growing and improving. Even if they continue to require supplemental oxygen, BPD infants may be discharged from the hospital if they are in stable condition on medication and if

the family and the baby's doctor agree that providing continuing care at home is best for the baby.

How Is BPD Treated?

There is no specific treatment for BPD.

There is no treatment that is specific for BPD. In the NICU supportive measures and *symptomatic treatment* are provided to help BPD babies breathe and give their lungs time to mature. The baby's lungs improve gradually through normal repair processes.

The treatment of BPD includes three components: therapy for RDS before BPD is confirmed, therapy after BPD is diagnosed, and home care. For infants who show signs and symptoms of RDS but who are not yet diagnosed with BPD treatment may include:

- surfactant administration to improve lung aeration,
- mechanical ventilators to make up for respiratory failure,
- supplemental oxygen to insure that the baby has enough oxygen in its blood,
- careful control of fluids to avoid pulmonary *edema* (accumulation of fluid in the lungs),

A nurse manages an infant with BPD who requires mechanical ventilation, supplemental oxygen, pharmacologic therapy and intravenous feeding.

- treatment for *patent ductus arteriosus*, a circulatory problem sometimes found in premature infants.
- giving the baby medicines that improve air flow in and out of the lungs, and
- feedings and appropriate supplemental formula to prevent malnutrition.

Once the diagnosis of BPD is confirmed the following treatments are continued in the NICU:

- continued mechanical ventilation and supplemental oxygen to overcome respiratory failure and maintain blood oxygen levels,
- *bronchodilator* medications to improve airflow in the lungs,
- *corticosteroids* and other medicines to reduce swelling and inflammation of *airways*,
- fluid restriction and *diuretics* to decrease water accumulation in the lungs
- *antibiotics* to control infections,
- intravenous feeding of needed nutrients, and
- physical therapy to improve muscle performance and to help the lungs expel *mucus*.

Scientists are working to develop new drugs and methods to prevent, lessen, or repair the lung injury that is seen with BPD. Some of the areas of research include:

- improving respirators so that fewer complications of positive pressure ventilation occur,
- using drugs to protect premature lungs from injury, or speed healing, and
- developing new drugs that improve lung function.

The best place for the baby's growth and development is at home with the family. It is important that the parents be loving and well-informed about the symptoms and treatment of BPD. These babies continue to have some respiratory symptoms for varying periods after leaving the hospital,

and they remain in fragile health. A primary care pediatrician should be available to provide acute, long-term, and preventive health care. In addition, nurses, respiratory and physical therapists, and social services may be needed.

What Are the Short- And Long-Term Consequences of BPD?

The symptoms that persist after the infant is discharged from the hospital vary. Babies with a history of BPD are more susceptible to respiratory infections and may continue to need low levels of supplemental oxygen. Some may remain dependent on a mechanical ventilator throughout early childhood.

Risk of developmental handicaps in BPD survivors is quite small but problems with lung function may persist into adulthood.

BPD survivors are at higher risk of complications after the usual childhood infections. As a precaution, hospital care may be recommended when a BPD baby becomes ill with a respiratory infection.

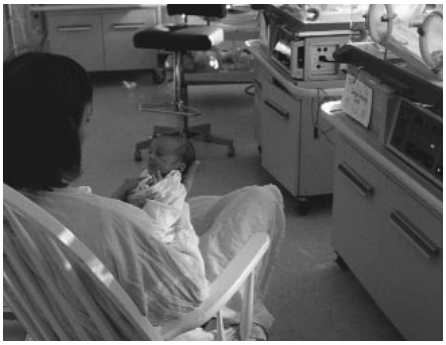
Babies who survive BPD grow more slowly than normal. This delayed growth continues into their second year of life. They usually remain smaller than normal children of the same age. Their lung growth is almost complete at 8 years of age as in all children, but they may continue to have some problems with their lung function even when they are adults.

The outlook for growth and development of babies with BPD varies. It depends more on the effects of prematurity and acute respiratory failure, rather than BPD itself. In very severe cases there may be some long-term limitations. These might include abnormalities in coordination, gait and muscle tone, inability to tolerate exercise, vision and hearing problems, and learning disabilities. The risk of these problems varies greatly among individual patients but is actually quite small. Parents of BPD infants need not assume that their child has a high risk of such developmental handicaps. If they

should occur, however, parents and families can obtain information about these problems from their baby's doctors.

LIVING WITH BPD

An infant with BPD may spend several weeks or months in the NICU. This is a stressful period for the parents and the family. While the baby remains in the hospital the parents should visit as frequently as possible, to bond with the baby and help the infant recognize the voices and touch of its parents.



A mother holding her baby with BPD left for intensive care in the hospital.

Social service agency personnel may be needed to teach parents of a baby with BPD how to play with and care for their infant. It is not uncommon for concern about the baby's medical condition to interfere with the parents care-giving abilities. Continued monitoring of the BPD survivor's growth and nutritional needs throughout infancy and childhood by a pediatric nutritionist can be reassuring to parents.

The parents of BPD infants can take a number of other steps to help their infants recover and grow as normally as possible. These include:

- seeking medical help when the child shows any signs of respiratory infection, for example, irritability, fever, nasal congestion, cough, changes in breathing pattern, wheezing;
- limiting the exposure of the infant to infections by avoiding the use of large day-care settings and crowds;
- protecting the baby from exposure to cigarette smoke and other respiratory irritants in the air; and

DEVELOPMENT OF THE NEONATAL LUNG

Before birth the respiratory needs of the *fetus* are met by the mother as her blood circulates through the *placenta*. The fetal lung, regardless of its stage of development, does not play a role in gas exchange. The fully developed, amniotic fluid-filled fetal lung is ready at the instant of birth to absorb the fluid and assume its own respiratory functions. The gestational age of the newborn determines the degree to which the infant's lungs are ready to function. Most neonatal deaths occur because the infant's lungs are not fully developed and are not ready to make the prompt transition necessary for survival outside of the womb.

The alveoli, the tiny air sacs in the lung where carbon dioxide and oxygen are exchanged, develop between 16 and 24 weeks of gestation. During this time capillaries grow in the lung tissue. Blood that is ready for gas exchange will flow through these capillaries. This period is called the canalicular stage of lung development. It is also marked by the appearance of the primitive *acinus*, the future lung tissue responsible for gas exchange.

The cells that line the alveoli are called epithelial cells. During this period of *prenatal* development these epithelial cells begin to change into type I and type II cells. After birth gas exchange occurs across the type I epithelial cells which are a flattened version of type II cells; type I cells form the air/blood barrier. Type II epithelial cells become the sites for surfactant production. Significant surfactant production does not normally occur until 34 weeks of gestation.

Toward the end of this developmental stage, respiration can take place because gas exchange can occur and some surfactant is present to keep the alveoli open. As a consequence, babies born at the end of the canalicular stage have a chance to survive. At this gestational age, however, their lungs are vulnerable to damage from infection, toxins, mechanical ventilation, and excessive amounts of oxygen. Oxidant radicals, a very reactive form of oxygen, are especially injurious since antioxidant defenses which normally protect the lung from the toxic effects of oxygen are not yet present in the immature lung.

HOW BPD DEVELOPS

Many substances that cause inflammation have been associated with the development of BPD. Magnesium, selenium, and copper deficiencies have also been suggested as possible factors. The specific roles of most of these agents in causing BPD have not yet been defined. When the neonatal lung is exposed to a damaging stimulus, the following events are believed to take place, resulting in BPD:

- **delayed development of the alveoli,**
- **inflammation and edema,**
- **disruption of surfactant function,**
- **infection,**
- **persistence of alveolitis (inflammation of the alveoli), and**
- **airway changes.**

During the first 1 to 2 weeks after the onset of BPD, the hyaline membrane resolves. Between 2 and 4 weeks a variety of abnormal conditions may appear. These include formation of fibrous tissue in the connective tissue of the lung and around the alveoli, blockage of airflow due to inflammation in the bronchioles, pulmonary *emphysema* (enlargement or destruction of alveoli), pulmonary hypertension (high blood pressure in the lung arteries), *pneumothorax* (accumulation of air in the spaces around the lung), patent ductus arteriosus, pulmonary infections, bronchial hyperreactivity, and small airway disease.

The most significant abnormal changes in the lungs of BPD infants appear to take place in the terminal bronchioles and alveolar ducts; the result is greatly reduced gas exchange. Other changes include loss of the cells that line the upper and lower airways, extensive scar formation, and collapse of the airways.

Respiratory failure generally results from severe airflow obstruction caused by swelling of the airway walls due to edema or plugging of the airways by mucus. Pulmonary hypertension contributes to the seriousness of the baby's condition. *Cor pulmonale* and systemic *hypertension* are some of the cardiovascular complications of BPD. Hypercalciurea (an excess of calcium in the urine), osteopenia (decrease in bone mass), and renal calcifications (kidney stones) related to nutritional problems and adverse effects of various treatments can also occur with BPD.

PROGRAMS OF THE NHLBI ON BPD

The NHLBI is promoting studies of how the normal lung develops and how its arrested or altered development leads to BPD. The goal of these efforts is to define the cellular events and reactions that occur as BPD develops (pathogenesis) and to understand the abnormal lung function (pathophysiology) of BPD. Research scientists are seeking ways to promote lung maturation and to forestall or minimize the consequences of lung prematurity at birth, to design more effective treatment methods for BPD, and to prevent prematurity. Some highlights of the NHLBI-supported research accomplishments are the following:

- An NIH Consensus Development Conference held in 1994 concluded that antenatal administration of corticosteroids to women at risk of premature delivery enhances fetal maturation and reduces mortality, respiratory distress, and intraventricular hemorrhage (bleeding into the brain) in preterm infants. Implementation of this recommendation has resulted in a substantial decrease in neonatal morbidity and mortality, as well as substantial savings in healthcare costs.
- The Collaborative Program for Research in BPD, established by the NHLBI in 1994, is supporting a multi-institutional consortium of interactive research grants which enables established investigators with different backgrounds and expertise to undertake a collective study of lung maturation using a baboon model of BPD. A major goal of this program is to develop strategies for both prevention and treatment of BPD. The baboon model is facilitating studies that are not possible in human patients.

- The gene for Thyroid Transcription Factor-1 (TTF-1) has been identified. TTF-1 appears to regulate lung formation during gestation. Some studies show that TTF-1 is shut off following lung injury in animals as well as in infants with BPD. This finding suggests that functional failure of TTF-1 may be responsible for the abnormal lung repair that accompanies BPD.
- Administration of retinoic acid, a derivative of vitamin A, has been shown to induce the formation of alveolar structures in newborn rats. Extrapolated to premature or newborn infants, this finding could provide a rationale for the suggested use of vitamin A in the management of BPD.
- Pulmonary neuroendocrine cells (PNECs) and the peptides they produce (for example, bombesin-like peptide, BLP) influence bronchial and vascular smooth muscle contractility, glandular secretion, and lung growth and development. Hyperplasia (abnormal increases in the number) of PNECs seems to be associated with airway epithelial regeneration during transition to BPD. Abnormally excessive secretion of BLP may be responsible for the peribronchial fibrosis, obliterative bronchiolitis, and hypertrophy of peribronchial smooth muscle in BPD. Urinary BLP levels were shown to increase during the first 24 to 72 hours after birth in premature baboons who go on to develop BPD. Furthermore, anti-bombesin *antibodies* can prevent or reduce the pathological changes that are characteristic of BPD in baboons.

- making sure that the baby and its siblings receive all routine *immunizations*. Some doctors now recommend shots to protect against infection with RSV (respiratory syncytial virus) which causes bronchiolitis.

Can BPD Be Prevented?

At present, the only practical way to prevent BPD is to eliminate high risk pregnancies that result in low birth weight babies. Programs of regular prenatal care for women at high risk of early delivery have been shown to lower the incidence of premature babies.

Scientists are studying ways to better understand the processes involved in premature labor and its prevention. In addition, research is being conducted on how to prevent or lessen the adverse effects that result when birth occurs before the lungs are mature. Ways are being sought to accelerate the process of lung maturation in infants at high risk of developing RDS and BPD. Providing corticosteroids to women at risk of premature delivery reduces infant mortality and decreases the incidence of RDS.

What Are the Healthcare Costs of BPD?

Infants with BPD need intensive hospital care for an average of 120 days. In 1990, the cost of caring for these infants was more than $170,000. These infants may also require home oxygen therapy for an average of 92 days. This cost is estimated to be more than $5,000 per child per year (1990 costs). However, if the infant were hospitalized during this period, the comparable cost would be $45,000 to $50,000. The overall costs of treating infants with BPD in the United States are estimated to be $2.4 billion. This amount is second only to the costs for treating asthma and far exceeds the cost of treating cystic fibrosis.

For More Information

The NHLBI Information Center is a service of the National Heart, Lung, and Blood Institute (NHLBI) of the National Institutes of Health. The Information Center provides information to health professionals, patients, and the public about the treatment, diagnosis, and prevention of heart, lung, and blood diseases. Contact the NHLBI or visit our home page for more information.

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Bethesda, MD 20824-0105
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The following organizations may have additional information on BPD:

American Lung Association
1740 Broadway
New York, NY 10019
Telephone: (212) 315-8700
e-mail: info@lungusa.org
Home page: http://www.lungusa.org

National Organization for Rare Diseases, Inc. (NORD)
P.O. Box 8923
New Fairfield, CT 06812-8923
Telephone: (203) 746-6518; or (800) 999-6673
e-mail: orphan@nord-rdb.com
Home page: http://www.nord-rdb.com/~orphan

GLOSSARY

Acinus: The berrylike ending of a tiny airway in the lung, where the alveoli (air sacs) are located.

Acute: Severe or with sudden onset and short timespan.

Airways: Tubes that carry air into and out of the lungs.

Alveoli: Tiny sac-like air spaces in the lung where carbon dioxide and oxygen are exchanged.

Amniotic fluid: The liquid that surrounds and cushions the fetus in its mother's womb.

Antibiotic: A drug that kills or inhibits the growth of bacteria.

Antibodies: Specific proteins produced by the body's immune system that bind with foreign proteins (antigens).

Artery: A blood vessel that carries blood from the heart to the rest of the body.

Asthma: Respiratory condition caused by narrowing of the airways; symptoms include recurrent attacks of wheezing, coughing, shortness of breath, and labored breathing.

Bronchiole: The smaller airways of the lungs.

Bronchiolitis: Inflammation of the bronchioles, usually caused by a viral infection.

Bronchodilator: A drug that relaxes the smooth muscles of the airways and relieves constriction of the bronchi.

Bronchopulmonary: Pertaining to the lungs and air passages.

Capillaries: The tiniest blood vessels; capillary networks connect the arterioles (the smallest arteries) and the venules (the smallest veins).

Cell: Basic subunit of every living organism; the simplest unit that can exist as an independent living system.

Chronic: Of long duration; frequently recurring.

Cor pulmonale: Heart disease that results from resistance to the passage of blood through the lungs; it often leads to right heart failure.

Corticosteroids: Drugs that mimic the action of a group of hormones produced by adrenal glands; they are anti-inflammatory and act as bronchodilators.

Cyanosis: Bluish color of the skin due to insufficient oxygen in the blood.

Cystic fibrosis: A serious genetic disease of excretory glands, affecting lungs and other organs; it causes production of very thick mucus that interferes with normal digestion and breathing.

Diuretic: A drug that promotes the excretion of salt and water by the kidney.

Duct: A passage or tube with well-defined walls for the passage of air or liquids.

Dysplasia: Abnormal development or growth.

Edema: Abnormal accumulation of fluid in body tissues.

Emphysema: Chronic lung disease in which there is permanent destruction of alveoli.

Fetus: Unborn offspring from 7 or 8 weeks after conception until birth.

Gas exchange: Primary function of the lungs; transfer of oxygen from inhaled air into the blood and of carbon dioxide from the blood into the lungs.

Genetic: Inherited through genes passed on by one or both parents.

Gestation period: The period of development of the young from the time of conception until birth.

Hyaline membrane disease: A respiratory disease of newborns, especially premature infants, in which a membrane composed of proteins and dead cells forms and lines the alveoli making gas exchange difficult or impossible.

Hypertension: High blood pressure.

Immunization: Protection from disease by administering vaccines that induce the body to form antibodies against infectious agents.

Inflammation: Response of the body tissues to injury; typical signs are swelling, redness, and pain.

Mechanical ventilation: Use of a machine called a ventilator or respirator to improve the exchange of air between the lungs and the atmosphere.

Membrane: Thin, flexible film of proteins and lipids that encloses the contents of a cell; it controls the substances that go into and come out of the cell. Also, a thin layer of tissue that covers the surface or lines the cavity of an organ.

Mucus: A thick fluid produced by the lining of some organs of the body.

Neonatal period: The first 4 weeks after birth.

Neonatologist: Doctor who specializes in treating the diseases and disorders of newborn babies.

Oxygen: Colorless odorless gas that makes up about 20 percent of the air we breathe; it is essential to life because it is used for the chemical reactions that occur in the cells of the body.

Patent ductus arteriosus: Abnormal persistence of the opening in the arterial duct that connects the pulmonary artery to the descending aorta; this opening normally closes within 24 hours of birth.

Pathogenesis: The cellular events and reactions that occur in the development of disease.

Pathophysiology: Altered functions in an individual or an organ due to disease.

Placenta: The special tissue that joins the mother to her fetus; it provides the fetus with oxygen, water, and nutrients (food) from the mother's blood and secretes the hormones necessary for successful pregnancy.

Pneumonia: Inflammation of the lungs.

Pneumothorax: Accumulation of air or gas in the space between the lung and chest wall, resulting in partial or complete collapse of the lung.

Positive pressure ventilation: Provision of oxygen under pressure by a mechanical respirator.

Prenatal: Occurring before birth.

Progressive: Increasing in severity.

Pulmonary: Pertaining to the lungs.

Pulmonary hypertension: Abnormally high blood pressure in the arteries of the lungs.

Respiration: Process of exchanging oxygen from the air for carbon dioxide from the body; includes the mechanical process of breathing, gas exchange, and oxygen and carbon dioxide transport to and from the cells.

Respiratory distress syndrome: A lung disease that occurs primarily in premature infants; the newborn must struggle for each breath and blueing of its skin reflects the baby's inability to get enough oxygen.

Respiratory failure: Inability of the lungs to conduct gas exchange.

Retina: The inner layer of tissue at the back of the eye that is sensitive to light.

Risk factors: Habits, traits, or conditions in a person or in the environment that are associated with an increased chance (risk) of disease.

Surfactant: Fluid secreted by the cells of the alveoli that reduces the surface tension of pulmonary fluids; it contributes to the elastic properties of pulmonary tissue.

Symptom: Any indication of disease noticed or felt by a patient; in contrast, a sign of an illness is an objective observation.

Symptomatic treatment: Therapy that eases symptoms without addressing the cause of disease.

Ventilation: Exchange of air between the lungs and the atmosphere so that oxygen can be exchanged for carbon dioxide at the alveoli.

Ventilator: A breathing machine that is used to treat respiratory failure by promoting ventilation; also called a respirator.

Wheezing: Breathing with a rasp or whistling sound; a sign of airway constriction or obstruction.

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U.S. DEPARTMENT OF HEALTH AND
HUMAN SERVICES

Public Health Service
National Institutes of Health
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